



Colonoscopy as a High Yielding Diagnostic Tool for per Rectum bleeding – An Overview

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ABSTRACT

Per rectum (PR) bleeding is a common cause of hospitalization and continues to be a problem for physicians. PR bleeding is defined as bleeding emanating from a source distal to the ligament of Treitz. Although bleeding stops spontaneously in 80% cases, 25% risk of re-bleeding persists along with a difficulty of identifying the bleeding source. Patients with major hemorrhage/ongoing bleed require rapid diagnosis and intervention to achieve hemostasis. Refinement of Endoscopic techniques has greatly improved the diagnosis and treatment of gastrointestinal bleeds. Colonoscopy is considered to be the primary mode of diagnosis, risk analysis and treatment of few common causes of colonic bleeding. The following review discusses the etiology of PR bleeds and reviews its colonoscopic diagnosis and treatment, along with the pros and cons of colonoscopy.

Key Words: Per Rectum Bleeding, Colonoscopy, LGIB

INTRODUCTION

Gastrointestinal (GI) bleeding refers to any form of hemorrhage/blood loss occurring in the gastrointestinal tract, a passage ranging from mouth to anus, and is divided into:

1. Upper GI bleeding (UGIB): Includes bleeding in esophagus, stomach, or initial part of the small intestine.
2. Lower GI bleeding (LGIB): Includes bleeding in remaining of the small intestine, large intestine, rectum, or anus.¹

PR bleeding refers to bleeding into enteric lumen originating distal to the ligament of Treitz.¹ It can be classified into occult, moderate or severe depending upon the amount of bleeding. Common clinical presentation includes passage of stools with bright red/maroon red blood, i.e., visible bleeding, iron deficiency anemia or positive fecal occult blood test.¹ Rectal bleeding is a common symptom occurring in about 20% of the general population, but only about 7 per 1000 patients per year seek medical opinion.² It is estimated that UGIB, LGIB and obscure bleeding account for 50%,

40% and 10% of GI bleeding respectively.³ Although LGIB stops spontaneously in 80% cases without needing hospitalization, identification of the bleeding source remains challenging and re-bleeding can occur in 25% of cases.^{4,5}

Etiology

Literature⁶ categorizes the etiologies of LGIB as follows:

Diverticular Disease

Colonic diverticulitis characterized by arterial bleeding occurring either at dome or neck of the diverticulum and presents as acute, painless hematochezia. It affects about two-third of the population above 80 years of age and the prevalence increases with age. These structures are the most common cause of acute LGIB (22% cases), considered to be the cause if no other source is found and identified by active bleeding or presence of a visible vessel/adherent clot.⁴ Bleeding is mostly (60% cases) observed in the left colon when diagnosed using colonoscopy but, is localized in right colon with angiography.^{6,7} Bleeding stops spontaneously in about

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80% of cases with a 25% cumulative risk of re-bleeding after 4 years.⁶ After endoscopic therapy, recurrence rate of bleeding is inconsistent; though Bloomfeld and colleagues reported an early recurrence in 38% of patients.⁸

Angiodysplasia

Angiodysplasias (*aka* vascular ectasias or angioectasias), though documented as cause of 30% LGIB cases, in reality cause only 3-12% of them.⁹ They majorly localize in the right hemicolon, often as multiple lesions with number increasing with age. The mucosal lesions are one millimeter to few centimeters in size and endoscopically appear circumscribed and red.¹⁰ They are seldom found during routine colonoscopy as most do not bleed, making patients asymptomatic warranting no therapy¹¹

Colitis

Colitis includes patients with inflammatory bowel disease (IBD), Crohn's disease, ulcerative, ischemic, infectious and pseudomembranous colitis.

Patients with IBD are usually characterized by non-life threatening bloody diarrhea. In one review, 50% patients with IBD caused intestinal hemorrhage experienced spontaneous halt in bleeding, while 35% experienced a re-bleeding episode;¹² a high recurrence rate warranting recommendation of semi-elective surgery after the first episode of severe GI bleeding secondary to IBD. Symptoms associated with Crohn's depend on the site, most common being terminal ileal or ileocecal disease.¹³ Ulcerative colitis, which affects only mucosa and sub-mucosa of the rectum and colon, cause only 2-8% of all LGIBs, out of which about 10% require emergent colectomies.^{4,14}

The cause of ischemic colitis is sudden, often temporary, reduction in mesenteric blood flow caused by hypotension/vasospasm. Patients experience sudden onset of mild abdominal pain usually followed by hematochezia/bloody diarrhea. Mostly bleeding ceases without interference; but colonic ischemia is linked with a high-risk mortality.¹⁵

In infectious colitis (including pseudo-membranous colitis), hemorrhage is rarely life threatening. Although any type of infectious colitis may cause hematochezia, the most common types are Enterohemorrhagic Escherichia, Salmonella, Histoplasma, and Cytomegalovirus.^{7,13}

Neoplasia

Neoplasia, though usually associated with microscopic blood in stool, is a lesion that can cause gross hematochezia when they wear down into underlying vessels caused by erosions of the luminal surface or polyps. Benign and malignant neoplasms of colon are the cause of LGIB in 10-20% of elderly cases.¹⁶ Bleeding is the initial presenting symptom in 26% of patients with colorectal neoplasms.¹⁷

Colorectal Cancer and Polyp

Carcinomas account for 2-9% of the LGIB cases.¹⁸ Erosions and ulceration on the surface of the tumor might bleed, and bleeding is often aggravated by the use of NSAIDs (Non-Steroidal Anti-Inflammatory Drugs). Carcinoma in the left colon leads to rectal bleeding, but often present as iron-deficiency anemia in the right colon. Colonic polyps are cited as the source of LGIB in 5-11% of patients and usually, only those larger than 1 cm bleed.¹⁴

Postpolypectomy Bleeding

Polypectomy procedure is associated with massive arterial bleeding which is caused by inadequate hemostasis of the blood vessel in polyp stalk.¹⁹ It is commonest complication of colonoscopy and is seen in 0.2-1.8% cases accounting for 2-8% cases of acute LGIB.²⁰ Delayed bleeding might occur up to 14 days after polypectomy.²¹

Anorectal Diseases

Chronic recurrent passage of red blood in small amounts is indicative of the bleeding source in anus, rectum or sigmoid. Hemorrhoids and chronic anal fissures are the most common bleeding source in young (< 40 years) patients with chronic LGIB.⁹ Hemorrhoids are the source of acute LGIB in 2-9% of patients.¹⁷ Acute LGIB from anal fissures is rare and usually ceases spontaneously. Fissures can be easily diagnosed by inspecting the anus but lesions need injecting of a local anesthetic for painless inspection.¹

Solitary rectal ulcers are also often associated with local ischemia accounting for rare bleeding, reported incidence being 18%. Rectal varices have a gray-blue color and cause severe bleeding in hypertension patients.²²

Radiation Proctitis

Radiation proctitis is a painful and persistent cause of LGIB in patients undergoing radiation therapy. It is characterized by permanently altered mucosal integrity and friable neovascularization which can bleed with minimal trauma. It presents 9-14 months after radiation therapy and cause hematochezia requiring pain management and endoscopic treatment.^{1,18}

Jejuno-Ileal Trauma

Jejuno-ileal diverticula are uncommon and cause only 1.1–2.3% of LGIB.²³ They commonly occur at the mesenteric border of jejunum in manifold. Though mostly asymptomatic, massive and recurrent GI bleeding is seen in about 3.4–8.1% of patients.²⁴

Meckel's Diverticulum

Meckel's Diverticulum is a rare cause of LGIB affecting only 2-3% of the population. This is a common congenital abnormality of small bowel caused by partial closure of the vitelline duct.²⁵ Gastric mucosa present in diverticulum

causes an ulcer in adjacent ileum, leading to painless rectal bleeding.

Aorto-Enteric Fistula

Aorto-enteric fistula is rare and can cause fatal hematochezia. They occur when compromised areas of bowel erode into the aortic wall. They can present up to after 14 years of surgery and are mostly seen in vascular surgery/trauma patients. Typically, patients present with self-limiting ‘herald’ bleed leading to massive hemorrhage and eventually death despite emergent surgery.^{5,17}

Dieulafoy's Lesion

Dieulafoy's lesion is a rare source of acute colonic bleeding and is not detected unless there is a visible bleed.²⁶ It is caused by an exposed artery that arises due to a minute mucosal defect.

Management of LGIB

Examination

Anorectal pathology is excluded by careful rectal examination of LGIB patients. Physical examination of patients is conducted to differentiate them based on severity of bleeding. There is no change in vital parameters of patient if blood loss is less than 200ml, but hemodynamic changes can be seen when blood loss is more than 800ml; shock if the blood loss is more than 1200-1500ml. Fatigue, dyspnea, pallor can also be seen. In LGIB patients, basic investigations like blood group, hemoglobin, prothrombin time, liver and kidney function test should be done.

Fresh frozen plasma and platelets play predominant role in treating patients with unbalanced coagulative profile and thrombocytopenia respectively. Vitamin k helps in reversing anti-coagulation though onset of action is delayed. Packed red cell are used in case of active bleeding. Age of patient and amount of bleeding decides the ideal hemoglobin concentration required. Healthy adult can be stable at hemoglobin concentration less than 7-8 g/dl (hematocrit < 20-25%), but this level increases risk in older patients.

Patients with stable vitals and no co-morbidity can be managed in wards. ICU management is required only in patients with active bleeding/unstable hemodynamics. Hemodynamics is crucial in determining patient prognosis; change in postural blood pressure even in absence of clinical feature suggests significant compromise. Detail abdominal examination should be done followed by cardiac and pulmonary exam.

Clinical Evaluation

Detailed patient history is taken about complaints of bleeding in past, radiation, polypectomy if any, coagulopathy, intake of aspirin/NSAIDs etc. History about amount, colour and duration of bleeding is also important. UGIB is considered if

there is history of melena/blood in nasogastric tube aspirates and can be associated with shock/hypotension.

Bloody diarrhea with abdominal pain is clinically significant, for in old patients it indicates ischemic colitis whereas in young patients it is suggestive of IBD. History of chronic renal failure and valvular heart disease in older patients indicate association with angiodysplasia, while history of aspirin/NSAIDs use is suggestive of ulceration throughout GI tract.²⁷

Diagnosis

Fibro-optic colonoscopy brought significant improvement in the diagnosis of colorectal problems in patients. Newer techniques such as colonoscopy, capsule endoscopy, double-balloon enteroscopy, single-balloon enteroscopy, push enteroscopy etc. play a key role in the diagnosis of LGIB. Of the above, colonoscopy is a highly sensitive, specific and relatively safe procedure with a low incidence of serious complications.¹⁸

Endoscopy

Flexible endoscopy is now considered primary route of diagnosis and treatment of acute/chronic colonic bleeding as the frequency of serious complications is as low as about 1 in 1,000 procedures.¹⁵ Patients are continuously monitored during urgent endoscopy using electrocardiogram and non-invasive measurement of oxygen saturation while hemodynamically unstable patients undergo volume resuscitation before endoscopy.¹⁸

Esophagogastroduodenoscopy is performed first in haemodynamically unstable patients to exclude an UGIB source, while colonoscopy is recommended as the first step in evaluation of acute LGIB.²⁸ The timing of colonoscopy after initial presentation varies among studies from 12-48 hours.⁷ Colonoscopy can determine the source and type of bleeding, along with identifying patients with ongoing hemorrhage or those at high risk of re-bleeding. Furthermore, endoscopic hemostasis can be performed, if necessary.

The documented diagnostic yield for urgent colonoscopy in acute LGIB is 75-97%.^{4,21} Thorough cleansing of the colon is recommended in acute LGIB patients as it facilitates endoscopic visualization, improves diagnostic yield, decreases perforation risk thereby presumably improving safety of the procedure.²⁰ For optimal purge of the colon, patient must ingest 3-6 lit of a polyethylene glycol-based solution. But, patients can tolerate about of 1-2 lit per hour, hence a prokinetic anti-emetic such as metoclopramide or administering the solution through a nasogastric tube is preferred.¹⁸

Colonoscopy can be started with the appearance of liquid discharge because diluted blood and clots can be aspirated/washed away. If the discharge becomes free of blood during preparation, endoscopic evaluation can be done on an

elective basis the following day. Cecum should be reached as hemicolon houses a significant number of bleeding sites while blood flow in terminal ileum indicates a proximal bleeding site. In patients with severe and ongoing bleeding, urgent colonoscopy must sometimes be carried out without purge.²⁰

Push enteroscopy enables visualization of about 50-120 cm of the proximal jejunum. Double-balloon enteroscopy can make the whole small intestine visible, especially if bidirectional enteroscopy is performed—that is, if the scope is introduced successively by mouth and anus. Using wireless video capsule endoscopy, the small bowel can be completely visualized in 80% of cases.¹⁸

Pros of Colonoscopy

A major advantage of colonoscopy over other management options is the potential for diagnosis and therapeutic intervention even in absence of ongoing bleeding unlike the radiographic alternatives including angiography, radionuclide scintigraphy and computed tomography, which need active ongoing bleed at the time of examination.²⁹ This is important as LGIB is often sporadic and may have slow bleeding mucosa.

The timing of colonoscopy also affects the diagnostic and therapeutic outcomes. Research indicates a better diagnostic and therapeutic yield with urgent colonoscopy, or colonoscopy within 12-24 hours of presentation in LGIB.³⁰ Endoscopic hemostasis is carried out post diagnosis based on source followed by appropriate treatment. Colonoscopy with or without intervention is considered a safe procedure for LGIB with a complication rate of 0.3% and 0.6% for regular and urgent colonoscopy respectively.³¹

Cons of Colonoscopy

Colon preparation is essential for complete caecum examination and includes cleansing the colon of stool and blood prior to colonoscopy. Unprepared colonoscopy leads to poor visualization increasing risk of perforation. Identification of subtle bleeding sites, often found among multiple lesions, requires removal of excess debris.

The preparation time (4 hours/more) required for colonoscopy preparations along with delay due to lack of nursing support and endoscopic facilities makes urgent colonoscopy logistically complicated.³⁰ Rapid preparation is associated with a risk of aspiration. Other potential risks in addition to bowel perforation include congestive heart failure secondary to volume overload, electrolyte abnormalities such as hyponatremia and aspiration pneumonia.³¹

Treatment

Endoscopic Hemostasis: Endoscopic treatment approach for LGIB comprises of injection, contact/non-contact thermal coagulation, mechanical devices such as metallic clips and

band ligation.²⁹ Other factors affecting the treatment modality are access to bleeding site, features of lesion and clinician's expertise.

Injection Therapy: Injection therapy is an inexpensive and straightforward method. Epinephrine (1:10,000 dilution) is usually used, causing vasoconstriction and physical compression of the vessel.²⁹ Addition of a sclerosant (eg, ethanolamine) seems to confer no additional advantage.²⁵

Thermal Coagulation: It is one of several hemostatic techniques. In bipolar (BICAP) and monopolar electro-coagulation, an electrical current from a probe heats up the tissue, whereas a heater probe directly delivers heat. All three probes induce coagulation upon contact. The BICAP probe shows an increase in energy deposition when greater pressure is applied, or when increased angulation causes an increase in tissue contact. For the heater probe, which has a non-stick Teflon coating, these associations have not been observed.³² BICAP and heater probe enable water jet irrigation through an opening on the probe tip.

Argon plasma coagulation: It transmits energy to the tissue without contact by means of ionized argon gas. The penetration depth is limited by the extent of tissue desiccation, whereas the coagulation depth depends on power setting, duration of application and distance between probe tip to target tissue. The perforation risk to colon seems to be almost non-existent.³³ In laser-mediated coagulation, the high-energy laser light causes vaporization of the tissue; deep penetration increasing perforation risk.

Mechanical devices: Metallic clips can be used to definitively and securely close bleeding lesions. Both reusable and single-use clipping devices are available.³⁴ The reusable device is usually less costly and available in different sizes, but requires a skilled assistant for loading and handling; after several uses, the rotation capability of the device becomes erratic. Single-use pre-loaded clipping devices are easy and quick to use. Ligation with rubber bands is used for bleeding hemorrhoids and bleeding rectal varices and, in certain circumstances, for treatment of focal lesions that are < 2 cm in diameter in non-fibrotic tissue. The amount of tissue suctioned into the cap before application of the rubber band must be carefully monitored owing to danger of full-thickness tissue entrapment and the subsequently increased risk of perforation.¹⁸

Interventional Angiography: The location and etiology of bleeding have important therapeutic implications for angiotherapy. Transcatheter embolization is an effective method of controlling GI hemorrhage. Embolization proximal to the mesenteric border of the colon was initially carried out via large catheters, which led to a significant rate of bowel infarction (13-33%).¹¹ However, the availability of microcatheters and embolization methods such as microcoils, gelfoam,

and polyvinyl alcohol particles renewed consideration for this technique.

Surgery: Most patients with LGIB will not require surgery, although it is the therapy of choice in patients with bleeding related to neoplasia.¹⁶ Surgery is also considered in patients for whom conservative therapies have failed but, identified bleeding source exists. In fulminant bleeding or patients with recurrent bleeding without source localization, surgery is the last resort. Blind segmental colectomy is associated with unacceptably high rates of morbidity, re-bleeding (75%) and mortality (up to 50%) and should therefore be avoided at all costs.³³

Whenever possible, intra-operative endoscopy should be carried out in such patients to clearly localize the bleeding source. Directed segmental resection is the treatment of choice because of its low morbidity, mortality (4%) and re-bleeding rate (6%).³³

DISCUSSION

Although colonoscopy for acute LGIB has been performed since 1970s, it has not been widely accepted in acute setting, partly due to fears about poor visibility in unprepared colon, and the potential for an increased risk of complications. However, with improvement in colonoscopy technology, including thinner and more flexible colonoscopes, studies demonstrated that colonoscopy is safe and effective in diagnosis of LGIB.^{3,4,8}

In randomized controlled trial on the use of urgent colonoscopy for evaluation of acute PR bleeding, 42% of patients had a definite diagnosis made when colonoscopy was performed within 8 hours of admission.³⁰ There is no consensus regarding the timing of urgent colonoscopy, which is determined by local resource availability. In literature, colonoscopy within 8 hrs, 12 hrs and 24 hrs are all reported as urgent colonoscopies.³¹ Green *et al* showed no difference in mortality, hospital stay, mean transfusion requirements, early or late re-bleeding or surgery when comparing urgent colonoscopy (within 8 hrs) to standard medical care (without colonoscopy but including angiography).³⁵

However, two other studies found that early colonoscopy (within 24 hrs) was associated with shorter hospital stay.³¹ In addition, Strate *et al* found that early colonoscopy resulted on significantly more therapeutic interventions.³¹ Although urgent colonoscopy has been performed without bowel preparation with reasonable diagnostic yield, rapid intake of oral solution using a balanced electrolyte ensures better visualization.³⁵

CONCLUSION

Colonoscopy is both diagnostic and therapeutic; thereby helping identify the site as well as cause of bleeding. Though colonoscopic screening is dangerous, expensive and needs a skilled endoscopist, the diagnostic yield is high in patients presenting with PR bleeding. It is precise and reliable in confirming diagnosis as it takes biopsy from growth. Also, frequent up-gradation in technology has demonstrated that colonoscopy is safe and effective in diagnosis and treatment of PR bleeds.

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Conflict of Interest

None

Abbreviations Used

PR	:	Per Rectum
GI	:	Gastro-Intestinal
UGIB	:	Upper Gastro-Intestinal Bleed
LGIB	:	Lower Gastro-Intestinal Bleed
IBD	:	Inflammatory Bowel Disease
NSAIDs	:	Non-Steroidal Anti-Inflammatory Drugs

REFERENCES

1. Davila RE, Rajan E, Adler DG, Egan J, Hirota WK, Leighton JA, et al. ASGE Guideline: the role of endoscopy in the patient with lower-GI bleeding. Gastrointest Endosc 2005;62:656–60.
2. Fijten GH, Muris JW, Starmans R, Knottnerus JA. The incidence and outcome of rectal bleeding in general practice. Fam Pract 1993;10(3):283-7.
3. Savides TJ and Jensen DM. Gastrointestinal Bleeding. In: Feldman M, Firsching S and Brandt LJ (eds), Sleisenger and Fordtran's. Gastrointestinal and Liver Disease. 9th ed. Elsevier Medicine; 2010.
4. Jensen DM, Machicado GA, Jutaba R, Kovacs Tao. Urgent colonoscopy for the diagnosis and treatment of severe diverticular hemorrhage. N Engl J Med 2000;342:78-82.
5. Imdahl A. Genesis and pathophysiology of lower gastrointestinal bleeding. Langenbecks Arch Surg 2001;386:1-7.

6. Lubis M, Zain LH. Etiology Profile of Lower Gastrointestinal Bleeding. *Indones J Gastroenterol Hepatol Dig Endosc* 2012;13(2):94-6.
7. Farrel JJ Friedman LS. Review article: the management of lower gastrointestinal bleeding. *Aliment Pharmacol Ther*. 2005;21:1281-98.
8. Bloomfeld RS, Rockey DC, Shetzline MA. Endoscopic therapy of acute diverticular hemorrhage. *Am J Gastroenterol*. 2001;96:2367-72.
9. Zuckerman GR, Prakash C. Acute lower intestinal bleeding. Part II: etiology, therapy, and outcomes. *Gastrointest Endosc*. 1999;49:228-38.
10. Richter JM, Christensen MR, Colditz GA, Nishioka NS. Angiodysplasia. Natural history and efficacy of therapeutic interventions. *Dig Dis Sci*. 1989;34:1542-6.
11. Fouch PG. Angiodysplasia of the gastrointestinal tract. *Am J Gastroenterol*. 1993;88:807-18.
12. Pardi DS et al. Acute major gastrointestinal hemorrhage in inflammatory bowel disease. *Gastrointest Endosc*. 1999;49:153-7.
13. Dent MT, Freeman AH, Dickinson RJ. Massive gastrointestinal bleeding in Crohn's disease. *J R Soc Med* 1985;78:628-9.
14. Robert JR, Sachar DB, Greenstein AJ. Severe gastrointestinal hemorrhage in Crohn's disease. *Ann Surg* 1991;213:207-11.
15. Strate LL, Ayanian JZ, Kotler G, Syngal S. Risk factors for mortality in lower intestinal bleeding. *Clin Gastroenterol Hepatol*. 2008;6:1004-10.
16. Boley SJ, DiBiase A, Brandt LJ, Sammartano RJ. Lower intestinal bleeding in the elderly. *Am J Surg* 1979;137:57-64.
17. Peura DA, Lanza FL, Gostout CJ, Fouch PG. The American College of Gastroenterology Bleeding Registry: preliminary findings. *Am J Gastroenterol* 1997;92(6):924-8.
18. Barnert J. Acute and chronic lower gastrointestinal bleeding. In: Messmann H editor. *Atlas of Colonoscopy*. Thieme, Stuttgart New York; 2006.p.118-42.
19. Kim HS et al. Risk factors for immediate postpolypectomy bleeding of the colon: a multicenter study. *Am J Gastroenterol*. 2006;101:1333-41.
20. Heldwein W et al. The Munich Polypectomy Study (MUPS): prospective analysis of complications and risk factors in 4000 colonic snare polypectomies. *Endoscopy* 2005;37:1116-22.
21. Sawhney MS, Salfiti N, Nelson DB, Lederle FA, Bond JH. Risk factors for severe delayed postpolypectomy bleeding. *Endoscopy* 2008;40:115-9.
22. Ganguly S, Sarin SK, Bhatia V, Lahoti D. The prevalence and spectrum of colonic lesions in patients with cirrhotic and noncirrhotic portal hypertension. *Hepatology* 1995;21:1226-31.
23. Maglinte DD, Chernish SM, DeWeese R et al. Acquired jejunoleal diverticular disease: subject review. *Radiology* 1986;158:577-80.
24. Yen HH, Chen YY, Yang CW et al. Diagnosis and management of jejunoleal diverticular haemorrhage: a decade of experience. *J Dig Dis* 2012;13:316-20.
25. Sagar J, Kumar V, Shah DK. Meckel's diverticulum: a systematic review. *J R Soc Med* 2006;99:501-5.
26. Raju GS, Gerson L, Das A et al. American Gastroenterological Association (AGA) institute technical review on obscure gastrointestinal bleeding. *Gastroenterology* 2007;133:1697-717.
27. Chan FK, Lanas A, Scheiman J, Berger MF, Nguyen H, Goldstein JL; Celecoxib versus omeprazole and diclofenac in patients with osteoarthritis and rheumatoid arthritis (CONDOR): a randomized trial. *Lancet*. 2010;376 (9736):173-9.
28. Eisen GM et al. An annotated algorithmic approach to acute lower gastrointestinal bleeding. *Gastrointest Endosc*. 2001;53:859-63.
29. Steer ML, Silen W. Diagnostic procedures in gastrointestinal hemorrhage. *N Engl J Med* 1983; 309(11):646-50.
30. Sakurai OT. Analysis of urgent colonoscopy for lower gastrointestinal tract bleeding. *Digestion* 2000;61(3):189-92.
31. Strate LL. Timing of colonoscopy; impact on length of hospital stay in patients with acute lower intestinal bleeding. *Am J Gastroenterol* 2003;98(2):317-22.
32. Swain CP et al. Which electrode? A comparison of four endoscopic methods of electrocoagulation in experimental bleeding ulcers. *Gut* 1984;25:1424-31.
33. Kwan V et al. Argon plasma coagulation in the management of symptomatic gastrointestinal vascular lesions: experience in 100 consecutive patients with long-term follow-up. *Am J Gastroenterol*. 2006;101:58-63.
34. Muhldorfer SM, Kekos G, Hahn EG, et al. Complications of therapeutic gastrointestinal endoscopy. *Endoscopy*. 1992;24:276-83.
35. Green BT, Rockey DC, Portwood G, Tarnsky PR. Urgent colonoscopy for evaluation and management of acute lower gastrointestinal hemorrhage; *Am J Gastroenterol* 2005;100(11): 2395-402.